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Cognitive, Emotional, and Social Processes in Psychosis: Refining Cognitive Behavioral Therapy for Persistent Positive Symptoms

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Psychosis used to be thought of as essentially a biological condition unamenable to psychological interventions. However, more recent research has shown that positive symptoms such as delusions and hallucinations are on a continuum with normality and therefore might also be susceptible to adaptations of the cognitive behavioral therapies found useful for anxiety and depression. In the context of a model of cognitive, emotional, and social processes in psychosis, the latest evidence for the putative psychological mechanisms that elicit and maintain symptoms is reviewed. There is now good support for emotional processes in psychosis, for the role of cognitive processes including reasoning biases, for the central role of appraisal, and for the effects of the social environment, including stress and trauma. We have also used virtual environments to test our hypotheses. These developments have improved our understanding of symptom dimensions such as distress and conviction and also provide a rationale for interventions, which have some evidence of efficacy. Therapeutic approaches are described as follows: a collaborative therapeutic relationship, managing dysphoria, helping service users reappraise their beliefs to reduce distress, working on negative schemas, managing and reducing stressful environments if possible, compensating for reasoning biases by using disconfirmation strategies, and considering the full range of evidence in order to reduce high conviction. Theoretical ideas supported by experimental evidence can inform the development of cognitive behavior therapy for persistent positive symptoms of psychosis.

Key words: schizophrenia/model/continuum/psychological interventions

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Introduction

> As an inpatient, I was in a psychiatric ward for people who seemed worn out by life. I was entertaining competing theories of why I was there. A part of me was aware that other people saw me as mentally ill and that I had become a social outcast.
>
> Rufus May[1]

Schizophrenia and related psychotic disorders create enormous burdens for individuals who suffer from them, for their carers, for the mental health services, and for society at large.[2] People with psychosis have always endured very poor social outcomes, including 80% unemployment rates.[3,4] Even worse, they have been stigmatized and misunderstood. The lifetime risk of committing suicide is 5%[5] with up to 13% showing moderate to severe suicidal behavior in a recent study.[6]

For most of the 20th century, scientific explanations of schizophrenia emphasized its otherness. The statements and experiences of people with the disorder were regarded as quintessentially incomprehensible.[7,8] Such formulations encouraged the conceptualization of schizophrenia as a distinct and distinguishable category and the postulation of a discreet biological causation. It also led to a focus on biological treatment at the expense of psychological interventions. Partly as a consequence, the symptoms of psychosis were seen primarily as the building blocks of diagnosis, rather than having an interesting and meaningful content.

Toward the end of the 20th century, it became increasingly apparent that the focus on biological mechanisms and treatment was restricting the possibilities of ameliorating the condition. Medication remains the first line of treatment. However, it is far from wholly effective, not only because 50% of people do not take their prescriptions reliably.[9] Many patients remain treatment resistant despite adequate doses of antipsychotic drugs, and side effects may impair consistent and optimal treatment.[10,11] Persistent positive symptoms such as hallucinations and delusions can be severely distressing and disruptive of daily functioning.

The ineffectiveness of intensive psychotherapy (which sometimes makes outcomes worse[12]) also contributed to an increasing pessimism. However, the 1990s saw the emergence of a paradigm shift, brought about by the

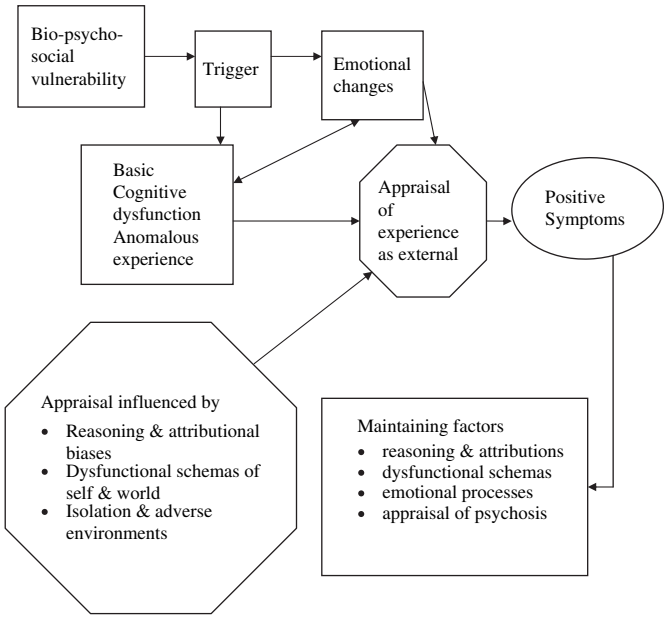

Fig. 1. A Cognitive Model of the Positive Symptoms of Psychosis (As Discussed in Garety et al).[14]

user movement,[1] new ideas about recovery,[13] and renewed interest in psychological treatments. Some years ago, we proposed a social and cognitive model of psychosis (figure 1) as a way of summarizing what was known and, hopefully, of guiding new research.[14] Since then, a considerable amount of research has illuminated social and psychological mechanisms in psychosis. Related to this has been increasing support for a view of psychosis as existing on a continuum with normal experience. These findings have between them begun a process of normalization, whereby psychotic experience is seen as less strange and more understandable. In this article, we use our model as an organizing principle to describe this new research.

A Cognitive Model of Psychosis[14]

Our cognitive model of psychosis conceptualizes the combination of factors that shape and maintain positive symptoms such as delusions and hallucinations. It incorporates the continuity of psychotic and nonpsychotic experiences and the idea of a biopsychosocial vulnerability which can be triggered by events. We posit that appraisal plays a central role in that it is not unusual experiences per se but a person's appraisal of them that can lead to symptoms. We think that emotional changes and low self-esteem are particularly important. We have also tried to incorporate findings that relate to adverse social environments. Unlike Broome et al,[15] we do not specify biological explanations, although these are implicit. We emphasize, as one route to symptom development, the importance of cognitive dysfunction such as information processing deficits which can lead to anomalous experiences. In contrast to Morrison et al,[16] we see this as distinguishing between psychosis and disorders such as anxiety or depression. Further, we suggest that reasoning biases play a particular role in symptom formation and their subsequent maintenance. Our research group have been able to test some of the putative pathways in our model, as indeed have other researchers. The most recent results are summarized below.

Studies of Psychosis As a Continuum—A Biopsychosocial Vulnerability

One of the consequences of moving away from a rigid categorical view of the symptoms of schizophrenia is that it reconfigures the problem of the threshold for recognizing the condition. We now think that the biopsychosocial vulnerability of psychosis is best conceived in terms of various criteria. In the late 1980s, evidence began to accumulate that the experiences described by patients, such as feelings of paranoia and hearing voices, were not confined to clinical groups. Bentall et al[17] showed that up to 25% of the normal population experienced hallucinations at least once, and Tien et al[18] in the general population found an annual incidence of 4–5% for hallucinations. Johns et al[19] showed that up to 4% of individuals in a population survey had these experiences. Freeman et al[20] have recently found that up to 30% of a nonclinical population have paranoid ideas. P. E. Bebbington, D. Freeman, C. Steel, J. Coid, E. Kuipers, T. Brugha, R. Jenkins, H. Meltzer, N. Singleton (unpublished data, 2006) have confirmed in a large population study that the normal population is also vulnerable to such experiences.

This overlap between clinical and nonclinical groups has been confirmed in other studies. Romme[21] was one of the first to publish in this area, with a study of voice hearers, 30% of whom experienced voices. In this group, the distinguishing feature of those in contact with mental health services was their level of distress. Davis et al[22] compared people with psychosis with evangelical Christian groups. The main difference was not their experience of voices but in the fact that the evangelical group felt more positive about them, while the clinical groups were more distressed. Similarly, Peters et al[23] found that when members of new religious movements such as Hare Krishna or Druids were compared with patients with psychosis, the former had similar levels of conviction in their beliefs but less distress. Van Os et al[24] and Hanssen et al[25] also showed that the differences between those with psychosis and "normals" were quantitative rather than qualitative and that distress contributed to patient status. Again, unusual experiences overlapped but reactions such as distress did not. Distress was consistently found in clinical rather than nonclinical groups and was much more likely to bring people to clinical services.

Research Into Individual Symptoms and Their Dimensions

Since the 1980s, Richard Bentall has maintained that schizophrenia is a "failed category" in terms of being able to predict specific treatment or outcomes consistently.[26] He has argued for "single symptom" research into delusions or hallucinations instead of research into schizophrenia as a whole. This argument has been persuasive, and much psychological research has concentrated on developing a clearer understanding of delusions,[27] paranoia,[28] and hallucinations.[29] This has allowed a clearer focus on the dimensions of individual symptoms, such as distress, conviction, and preoccupation, and has led to more targeted psychological intervention, for instance for paranoia.[30] Recently, genetic studies have also raised doubts about the utility of separate categories of schizophrenia and bipolar disorder.[31]

Emotional Changes in Psychosis

A diagnosis of schizophrenia has tended to discount the considerable amount of emotional disorder associated with it, in a manner that can also inform psychological treatment options. For instance, depression is often associated with schizophrenia, such that up to 40% of people with the latter also have clinical levels of depression,[32,33] accompanying low levels of self-esteem,[34] and a high risk of suicide[5]. In all, 30% fit the criteria for posttraumatic stress disorder,[35] 20% may have panic disorder,[36] 25% have evidence of obsessional compulsive disorder,[37] 40% have a dual diagnosis of comorbid substance misuse,[38] and 50% a comorbid personality disorder.[39,40] This comorbidity adversely affects outcome, with accompanying high inpatient bed use. It may also reflect on processes underlying the development of psychotic symptoms.

Our group is particularly interested in the impact of emotional changes on symptom formation and maintenance. Using a new version of a schema questionnaire,[41] we have found that the positive symptoms of psychosis, delusions and hallucinations, are associated with extreme negative evaluations of the self and others.[42] In corroboration of this possible pathway, Barrowclough et al[43] found that low self-esteem in patients was associated with negative evaluations (criticism) by carers and higher symptoms. Similarly, Krabbendam et al[44] have shown that depression contributes to the later development in delusions in people with preexisting anomalies of experience. Myin-Germeys and colleagues[45] demonstrated that fluctuations in positive symptoms of psychosis are associated with time-sampled changes in negative affect.

The Central Role of Appraisal

Birchwood[33] has found that appraisals of auditory hallucinations as powerful and controlling are linked to depressed mood. We have also found this for persecutory delusions.[46] Further, negative appraisals of symptoms, of self and of others relate to suicidal ideation and high alcohol intake.[47] Morrison et al[16] showed that, like people with phobic disorders, individuals with psychosis exhibit safety behaviors. We have recently confirmed this and have shown that they relate specifically to delusional persistence.[48,49] Along with other investigators, we have also looked at illness appraisals, using methods developed in health psychology. Lobban and colleagues have shown that these measures can be used in psychosis[50] and that patients make similar appraisals in both physical and mental health conditions. We also found that negative illness appraisals of psychosis were associated with distress.[51]

Cognitive Processes: Reasoning and Attributional Biases

The importance of reasoning biases in psychosis has been confirmed by recent research. Many of us hold with conviction ideas that do not have much basis in evidence, for instance, beliefs in astrology, alien beings, telepathy, or ghosts. A quarter of us act on the basis of beliefs in our star signs. Further, once we hold a strong belief, it is normal for us not to consider alternatives impartially, the so-called "confirmatory bias." For those with psychosis, it has been found that in addition to these normal biases, they tend to use less evidence before making a decision, the "jumping to conclusions" (JTC) reasoning bias,[27] which has particular relevance for delusional thinking. We have found that reasoning biases contribute differentially to delusional symptom dimensions, specifically to conviction.[52] In sample of 100 patients with psychosis, 50% showed a JTC reasoning bias, and this contributed to delusional conviction, whereas disturbed affect was linked to delusional distress.

JTC is found both in people with delusions and also in those in recovery from delusions.[53] Further, JTC is related to belief inflexibility and to an inability to generate alternative explanations for experiences.[54]

Bentall and colleagues[55] have also found evidence of "attributional biases," although this is now less well supported.[56] In particular, some people with persecutory delusions have an "externalizing bias," being more likely to attribute blame for negative events to external factors, particularly to other people. This contrasts markedly with the self-blame of people with depression.

Another elegant set of experiments supports the idea of "self-monitoring problems" in psychosis,[57,58] which can lead to hallucinations or delusions of control.[19,59] There is also evidence for what Hemsley has called "disruption to a sense of self"[60,61] in that poor use of contextual information can disrupt the ability to process ongoing experiences.[62] Kapur[63,64] has recently argued that abnormalities of dopaminergic activity are related to our understanding of the salience of experiences, and an

excess may form one of the routes to ideas of reference in acute episodes.

We also know that people with psychosis have well-established "cognitive deficits" in attention and working memory.[65] These are factors to bear in mind while offering interventions but may also contribute to symptom formation. Clancy et al[66] in an experimental investigation of individuals claiming to have experienced alien abduction found that these experiences were related both to a high rate of sleep paralysis and to false recall and recognition deficits.

Psychosis and the Social Environment: Triggering and Maintaining Events

A recent study reported that supportive social environments are associated with reduced positive symptoms of psychosis and that family support relates particularly to reduced hospital admissions up to 3 years after a first episode.[67] Aspects of adverse family environment can be tapped by the expressed emotion measure, which is well established as a predictor of relapse.[68] We have recently found that high levels of expressed emotion in carers relate to negative affect. In particular, in patients with a recent relapse of psychosis, criticism on the part of carers predicted anxiety but not more severe psychotic symptoms.[69]

It is becoming increasingly acknowledged that high rates of trauma and adversity occur before the onset of psychosis, often years before.[70–72] These studies confirm that bullying and sexual abuse are associated with negative self and other schemas and with positive symptoms such as persecutory delusions and hallucinations. There may be a particular relationship with hallucinations, although direct links between trauma and hallucinatory content were relatively rare.[73] Recent studies have shown links between specific attributes of recent events and the content of delusions and hallucinations in a first episode sample.[74]

We have hypothesized that trauma and adversity affect both information and emotional processing, leading to intrusions which are then misinterpreted and appraised as symptoms of psychosis.[41,75]

Virtual Reality Studies

Alongside the above studies, we have developed novel virtual reality paradigms in collaboration with Professor Mel Slater at University College London, UK. It is possible to immerse people in a virtual reality "cave" such that they are able to move around in virtual environments in real time. This allows for the control of an environment peopled with avatars (computer generated human figures that provoke emotional reactions in people in the same way that cartoons do). Within these environments (a library scene and an underground tube train), we have been able to show that nonclinical individuals may develop persecutory thoughts about avatars: eg, "they were telling me to go away.[76]" Anomalous experiences differentiated in normal samples between individuals who were just anxious and those who had persecutory ideas.[77] Preliminary work on prodromal samples suggests that such thinking is on a continuum, as predicted from earlier studies. We plan to extend this work into clinical samples with the intention that it will inform treatment approaches.

Therapeutic Approaches in Cognitive Behavior Therapy (CBT) for Positive Symptoms of Psychosis

Evidence Base

The first description of a cognitive behavioral approach to delusional ideas was provided by Beck,[78] who discussed his client's persecutory ideas in a case study. However, Beck did not pursue cognitive research in psychosis at that time, and the next reports were of case studies and uncontrolled studies in the 1970s and 1980s. The main evidence for CBT for psychosis comes from the United Kingdom and has been driven by 4 main research groups, associated particularly with the names of Garety, Kuipers, and Fowler in London and East Anglia, Tarrier and Bentall in Manchester, Kingdon and Turkington in Southampton and Newcastle, and Birchwood and colleagues in Birmingham. These groups published a series of randomized controlled trials on CBT for psychosis, which have now been the subject of several meta-analyses.[79–82] The last of these covered 14 randomized controlled trials (RCTs) ($N = 1484$). Studies have varied in their approach with some negative findings. Overall, there is an effect size of around 0.37 for CBT, with best effects on improvements in persistent positive symptoms. All trials include participants already on antipsychotic medication. Jones et al[81] call CBT "a promising but under-evaluated intervention." There is some evidence emerging for the value of early intervention services and for intervention in prodromal states, but there are few controlled trials of treatment at this stage.[83–87]

In the United Kingdom, the National Institute of Clinical Excellence published guidelines for the treatment of schizophrenia on the basis of its own review of the evidence in 2002.[88] These recommended that CBT be offered to those with persistent positive symptoms of psychosis for at least 10 sessions over at least 6 months. This guideline will be reviewed 2006/2007.

The Importance of Engagement

In line with our treatment manual[89] and those of other groups,[90–93] we have established that intervention to help people with persistent positive symptoms requires consideration of engagement and the formation of a therapeutic alliance, not just in early sessions but throughout

treatment (R. Rollinson, B. Smith, S. Steel, S. Jolley, J. Onwumere, D. Freeman, P. A. Garety, E. Kuipers, P. E. Bebbington, G. Dunn, M. Startup, D. Fowler, unpublished data, 2006). This entails therapists taking responsibility for keeping sessions nonaversive. It also requires that therapists remain aware of the possibility that the patient may have cognitive deficits such as poor concentration, poor memory, or poor executive and planning abilities and tailor interventions appropriately. Sessions may need to be kept short and be conducted flexibly; adhering to a rigid agenda may not be appropriate. It is also likely that during sessions, an individual may become suspicious of the therapist or otherwise distracted by cognitive distortions or intrusions such as voices. Therapists need to be alert to such probabilities to check them out and discuss them and, if possible, reassure the client, aiming in the process to reduce the ambiguities that can cause such disturbances. The aim of sessions initially, say the first five or six, is to conduct a thorough assessment and from this to collaborate in developing a formulation for distressing experience that can be shared by the therapist and the client. This then leads on to appropriate cognitive and behavioral interventions.

Managing Affect in Hallucination and Delusions

We know from the research described above that depression, anxiety, and social isolation are particularly associated with the development and maintenance of hallucinations and delusional distress.[42] In order to offer effective treatment, it is often helpful to consider assessing and treating the affect that usually precedes and accompanies distressing voices[44] and trying to interrupt cycles of low mood and anxiety. Clinically, it can be seen that introducing behavioral activity scheduling for low mood can not only improve affect but may also itself reduce isolation and improve social networks and social support. Monitoring and enhancing coping strategies for hallucinations can be helpful in reducing feelings of powerlessness and pessimism and improving the effectiveness of coping.[29] This in itself can sometimes reduce the frequency of voices.[94]

Reappraisal

Our model particularly focuses on the importance of reappraisal, especially of negative beliefs.[46] We know that delusional distress for instance is related to negative illness appraisals,[51] and it can be helpful to decatastrophize symptoms, to discuss and manage stigma and negative illness consequences, and to emphasize recovery models.[13] Trower et al,[29] in an RCT for individuals with command hallucinations, found it particularly helpful for individuals to deal directly with the consequences of the commands and to discover that the voices were not so powerful and controlling. Reduced distress was related to changes in cognitions of power and control.

It can also be useful to reappraise external attributions as internal states, so that individuals can recategorize paranoid ideas as internal worries, critical voices as mirroring self-blaming cognitions, or as memories of the critical voices of others.[43]

Work on Negative Schemas and to Manage and Reduce Stress

Given the research on abuse[72] and its effects on schemas and views about the self, other people, and the world,[41,95] together with the likely increase in intrusions that can result,[75] interventions may need to be attempted at the schema level. Work on reevaluating underlying schema and understanding how they continue to feed into the experience of negative voices or delusional ideas such as paranoia may be particularly useful for those with histories of abuse that continue to be triggered by everyday events and render people vulnerable to relapse.[96]

We know that isolated or critical social settings relate to dysfunctional affect, poor self-esteem, and increased positive symptoms.[69,43] Work on such aversive environments can help in reducing tensions, negotiating changes, managing disagreements, and building up more supportive and positive environments. If carers are involved, family interventions[97–100] may reduce relapses of positive symptoms and improve functioning.[88]

Dealing With High Conviction; Helping to Compensate for Reasoning Biases

We know that delusional conviction relates to JTC,[52] which is in turn linked to belief inflexibility and an inability to generate alternative explanations.[54] This suggests that for those people who hold their beliefs with strong conviction, it is important to work slowly on the acknowledgment that other explanations are credible. It may be necessary for the therapist to provide these because clients may not be able to generate them for themselves. Testing out new explanations to see if they are credible seems to be an important part of this process and should be attempted if possible.

Developing new strategies for clients to gather more information before making a decision can help with JTC biases and can become part of a more general style of "thinking the second thought" before coming to a conclusion. We also know that disconfirmation is less commonly used as a way of testing competing theories about what is happening: individuals usually employ confirmatory strategies.[54] Looking instead for instances, which do not fit in with predictions (eg, not everyone I passed was looking at me), can help to counter both confirmation and attributional biases.

Therapists should try to work collaboratively with clients to "see what happens" and encourage the gradual dropping of safety behavior and avoidance.[49] Clinical experience suggests that this has to be done slowly and in

small steps in order to reduce dysphoria-induced worsening of symptoms of psychosis. It is a good argument for not using "flooding" techniques with this population.

Conclusion

We are still developing CBT for the treatment of the distressing delusions and hallucinations associated with psychosis. There is some evidence that it can be helpful, particularly for persistent positive symptoms, and good evidence that it does not make things worse. It does not, for instance, increase suicide rates.[6] Our research group has been interested in specifying a cognitive model of such symptoms in order to test out putative mechanisms that elicit and maintain them. Results so far provide evidence for the influence of several of the hypothesized cognitive, emotional, and social factors. These are already improving our understanding of symptom dimensions and leading to a clearer rationale for intervention. Our current study, not yet completed, will allow us to investigate the mechanisms of any therapeutic change. This should in turn illuminate the processes involved in symptom formation and maintenance.

Untangling worries of things that might be,
Controlling and broadcasting all about me,
The tills in the shops or a panic alarm,
Untangling worries that may cause me harm.

Untangling voices for they cannot hurt
And I'm in control and I'm on the alert,
A voice has no body, it's all an illusion,
Untangling voices and all their confusion.

Extracts from an untitled poem by Wendy Baker, reproduced with the author's permission.

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References

1. May R. Routes to recovery from psychosis: the roots of a clinical psychologist. *Clin Psychol Forum.* 2000;146:6–10.
2. Knapp M, Mangalore R, Simon J. The global costs of schizophrenia. *Schizophr Bull.* 2004;30:279–293.
3. Marwaha S, Johnson S. Schizophrenia and employment: a review. *Soc Psychiatry Psychiatr Epidemiol.* 2004;39: 337–349.
4. Thornicroft G, Tansella M, Becker T, et al. The personal impact of schizophrenia in Europe. *Schizophr Res.* 2004;69: 125–132.
5. Palmer BA, Pankratz VS, Bostwick JM. The lifetime risk of suicide in schizophrenia: a re-examination. *Arch Gen Psychiatry.* 2005;62:247–253.
6. Tarrier N, Haddock G, Lewis S, Drake R, Gregg L. Suicide behaviour over 18 months in recent onset schizophrenic patients: the effects of CBT. *Schizophr Res.* 2006;83:15–27.
7. Jaspers K. *General Psychopathology (1912).* Hoenig J, Hamilton MW, trans-ed. Manchester, UK: Manchester University Press; 1963.
8. Berrios GE. *The History of Mental Symptoms: Descriptive Psychopathology Since the Nineteenth Century.* Cambridge, England: Cambridge University Press; 1996.
9. Kemp R, Hayward P, Applewhaite G, Everitt B, David A. Compliance therapy in psychotic patients: random controlled trial. *BMJ.* 1996;312:345–349.
10. Kane JM. Treatment-resistant schizophrenic patients. *J Clin Psychiatry.* 1996;57:35–40.
11. Lieberman JA, Stroup TS, McEvoy JP, et al. Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) Investigators. Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. *N Engl J Med.* 2005; 353:1209–1223.
12. Mueser KT, Berenbaum H. Psychodynamic treatment of schizophrenia: is there a future? *Psychol Med.* 1990;20: 253–262.
13. Borkin JR, Steffen JJ, Ensfield LB, et al. Recovery attitudes questionnaire: development and evaluation. *Psychiatr Rehabil J.* 2000;24:95–102.
14. Garety PA, Kuipers E, Fowler D, Freeman D, Bebbington P. A cognitive model of the positive symptoms of psychosis. *Psychol Med.* 2001;31:189–195.
15. Broome MR, Wooley JB, Tabraham P, et al. What causes the onset of psychosis? *Schizophr Res.* 2005;79:23–34.
16. Morrison AP. The interpretation of intrusions in psychosis: an integrative cognitive approach to hallucinations and delusions. *Behav Cogn Psychother.* 2001;29:257–276.
17. Bentall RP, Claridge G, Slade PD. The multidimensional nature of schizotypal traits: a factor-analytic investigation with normal subjects. *Br J Clin Psychol.* 1989;28: 363–375.
18. Tien AY. Distributions of hallucinations in the population. *Psychiatr Rehabil J.* 1991;26:287–292.
19. Johns LC, Rossell S, Ahmad F, et al. Verbal self-monitoring and auditory verbal hallucinations in patients with schizophrenia. *Psychol Med.* 2001;31:705–715.
20. Freeman D, Garety PA, Bebbington PE, et al. Psychological investigation of the structure of paranoia in a non-clinical population. *Br J Psychiatry.* 2005;186:427–435.
21. Romme MA, Honig A, Noorthoorn EO, Escher AD. Coping with hearing voices: an emancipatory approach. *Br J Psychiatry.* 1992;161:99–103.
22. Davis MF, Griffin MY, Vice S. Affective reactions to auditory hallucinations in psychotic, evangelical and control groups. *Br J Clin Psychol.* 2001;40:361–370.
23. Peters ER, Day S, McKenna J, Orbach G. The incidence of delusional ideation in religious and psychotic populations. *Br J Clin Psychol.* 1999;38:83–96.
24. Van Os J, Hverdoux H, Maurice-Tison S, et al. Self-reported psychosis-like symptoms and the continuum of psychosis. *Soc Psychiatry Psychiatr Epidemiol.* 1999;34:459–463.
25. Hanssen M, Peeters F, Krabbendam L, Radstake S, Verdoux H, van Os J. How psychotic are individuals with non-psychotic disorders? *Soc Psychiatry Psychiatr Epidemiol.* 2003;38:149–154.
26. Bentall RP. *Madness Explained: Psychosis and Human Nature.* London, England: Penguin; 2003.
27. Garety PA, Hemsley DR. *Delusions: Investigations Into the Psychology of Delusional Reasoning.* Oxford, England: Oxford University Press; 1994.

28. Freeman D, Garety PA. A cognitive model of persecutory delusions. In: Freeman D, Garety PA, eds. *Paranoia: The Psychology of Persecutory Delusions.* Maudsley Monograph No. 45. Hove, England: Psychology Press; 2004:115–135.
29. Trower P, Birchwood M, Meaden A, Byrne S, Nelson A, Ross K. Cognitive therapy for command hallucinations: randomised controlled trial. *Br J Psychiatry.* 2004;184: 312–320.
30. Freeman D, Freeman J, Garety P. *Overcoming Paranoid and Suspicious Thoughts. A Self-Help Guide Using Cognitive Behavioural Techniques.* London, England: Constable Robinson; 2006.
31. Craddock N, O'Donovan MC, Owen MJ. Genes for schizophrenia and bipolar disorder? Implications for psychiatric nosology. *Schizophr Bull.* 2006;32:9–16.
32. Sands JR, Harrow M. Depression during the longitudinal course of schizophrenia. *Schizophr Bull.* 1999;25:157–171.
33. Birchwood M. Pathways to emotional dysfunction in first-episode psychosis. *Br J Psychiatry.* 2003;182:373–375.
34. Freeman D, Garety P, Fowler D, et al. The London East Anglia RCT of CBT for Psychosis IV: self esteem and persecutory delusions. *Br J Clin Psychol.* 1998;37:415–430.
35. Mueser KT, Goodman LB, Trumbetta SL, et al. Trauma and posttraumatic stress disorder in severe mental illness. *J Consult Clin Psychol.* 1998;66:493–499.
36. Turnball G, Bebbington P. Anxiety and the schizophrenic process: clinical and epidemiological evidence. *Soc Psychiatry Psychiatr Epidemiol.* 2001;36:235–243.
37. Berman I, Kalinowski A, Berman SM, Lengua J, Green AI. Obsessive and compulsive symptoms in chronic schizophrenia. *Compr Psychiatry*. 1995;36:6–10.
38. Scott H, Johnson S, Menezes P, et al. Substance abuse and risk of aggression and offending among the severely mentally ill. *Br J Psychiatry.* 1998;172:345–350.
39. Keown P, Holloway F, Kuipers E. The prevalence of personality disorders, psychotic disorders and affective disorders amongst the patients seen by a community mental health team in London. *Soc Psychiatry Psychiatr Epidemiol.* 2002;37:225–229.
40. Keown P, Holloway F, Kuipers E. The impact of severe mental illness, comorbid personality disorders and demographic factors on psychiatric bed use. *Soc Psychiatry Psychiatr Epidemiol.* 2005;40:42–49.
41. Fowler DG, Freeman D, Smith B, et al. The brief core schema scales (BCSS): psychometric properties and associations with paranoia, depression and grandiosity in non-clinical and psychosis samples. *Psychol Med.* 2006;36: 749–759.
42. Smith B, Fowler DG, Freeman D, et al. Emotion and psychosis: direct links between depression, self-esteem, negative schematic beliefs and delusions and hallucinations. *Schizophr Res.* In press.
43. Barrowclough C, Tarrier N, Humphreys L, Ward J, Gregg L, Andrews B. Self-esteem in schizophrenia: relationships between self-evaluation, family attitudes, and symptomatology. *J Abnorm Psychol.* 2003;112:92–99.
44. Krabbendam L, Myin-Germeys I, Hanssen M, et al. Development of depressed mood predicts onset of psychotic disorder in individuals who report hallucinatory experiences. *Br J Clin Psychol.* 2005;44:113–125.
45. Myin-Germeys I, van Os J, Schwartz J, Stone A, Delespaul P. Emotional reactivity in daily life stress in psychosis. *Arch Gen Psychiatry.* 2001;58:1137–1144.
46. Green CEL, Garety PA, Freeman D. Content and affect in persecutory delusions. *Br J Clin Psychol.* In press.
47. Fialko L, Freeman D, Bebbington, et al. Understanding suicidal ideation in psychosis: findings from the Psychological Prevention of Relapse in Psychosis (PRP) Trial. *Acta Psychiatr Scand.* In press.
48. Freeman D, Garety PA. Connecting neurosis and psychosis: the direct influence of emotion on delusions and hallucinations. *Behav Res Ther.* 2003;41:923–947.
49. Freeman D, Garety P, Kuipers E, Fowler D, Bebbington PE, Dunn G. Acting on persecutory delusions: the importance of safety seeking. *Behav Res Ther.* In press.
50. Lobban F, Barrowclough C, Jones S. The impact of beliefs about mental health problems and coping on outcome in schizophrenia. *Psychol Med.* 2004;37:1165–1174.
51. Watson PWB, Garety PA, Weinman J, et al. Emotional dysfunction in schizophrenia spectrum psychosis: the role of illness perceptions. *Psychol Med.* 2006;36:761–770.
52. Garety P, Freeman D, Jolley S, et al. Reasoning, emotions and delusional conviction in psychosis. *J Abnorm Psychol.* 2005;114:373–384.
53. Peters E, Garety P. Cognitive functioning in delusions: a longitudinal analysis. *Behav Res Ther.* 2006;44:481–514.
54. Freeman D, Garety PA, Fowler D, Kuipers E, Bebbington PE, Dunn G. Why do people with delusions fail to choose more realistic explanations for their experiences? An empirical investigation. *J Consult Clin Psychol.* 2004;72:671–680.
55. Bentall R, Kinderman P, Kaney S. The self, attributional processes and abnormal beliefs: towards a model of persecutory delusions. *Behav Res Ther.* 1994;32:331–341.
56. Jolley S, Garety P, Bebbington P, et al. Attributional style in psychosis—the role of affect and belief type. *Behav Res Ther*. In press.
57. Frith CD. *The Cognitive Neuropsychology of Schizophrenia.* Hove, England: Lawrence Erlbaum Associates; 1992.
58. Frith CD. The neural basis of hallucinations and delusions. *C R Biol.* 2005;328:169–175.
59. Blakemore SJ, Oakley DA, Frith CD. Delusions of alien control in the normal brain. *Neuropsychologia*. 2003; 41:1058–1067.
60. Hemsley DR. The disruption of the 'sense of self' in schizophrenia: potential links with disturbances of information processing. *Br J Med Psychol.* 1998;71:115–124.
61. Hemsley DR. The schizophrenic experience: taken out of context? *Schizophr Bull.* 2005;31:43–53.
62. Barch D, Mitropoulou V, Harvey PD, New AS, Silverman JM, Siever LJ. Context-processing deficits in schizotypal personality disorder. *J Abnorm Psychol.* 2004;113:556–568.
63. Kapur S. Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *Am J Psychiatry*. 2003;160:13–23.
64. Kapur S, Arenovich T, Agid O, Zipursky R, Lindborg S, Jones B. Evidence for onset of antipsychotic effects within the first 24 hours of treatment. Multicenter study. Randomized controlled trial. *Am J Psychiatry*. 2005;162:939–946.
65. Joyce E. Origins of cognitive dysfunction in schizophrenia: clues from age at onset. *Br J Psychiatry*. 2005;186:93–95.
66. Clancy SA, McNally RJ, Schacter DL, Lenzenweger MF. Memory distortion in people reporting abduction by aliens. *J Abnorm Psychiatry*. 2002;111:455–461.
67. Norman RMG, Malla AK, Manchanda R, Harricharan R, Takhar J, Northcott S. Social support and three-year

symptom and admission outcomes for first episode psychosis. *Schizophr Res.* 2005;80:227–234.

68. Bebbington P, Kuipers L. The predictive utility of EE in schizophrenia: an aggregate analysis. *Psychol Med.* 1994; 24:707–718.
69. Kuipers E, Bebbington P, Dunn G, et al. Influence of carer expressed emotion and affect on relapse in non-affective psychosis. *Br J Psychiatry*. 2006;188:173–179.
70. Krabbendam L, Myin-Germeys I, De Graaf R, et al. Dimensions of depression, mania and psychosis in the general population. *Psychol Med.* 2004;34:1177–1186.
71. Bebbington PE, Bhugra D, Brugha T, et al. Psychosis, victimisation and childhood disadvantage: evidence from the second British National Survey of Psychiatric Morbidity. *Br J Psychiatry*. 2004;185:220–226.
72. Read J, van Os J, Morrison AP, Ross A. Childhood trauma, psychosis and schizophrenia: a literature review with theoretical and clinical implications. *Acta Psychiatr Scand.* 2005;112:330–350.
73. Hardy A, Fowler D, Freeman D, et al. Trauma and hallucinatory experience in psychosis. *J Nerv Ment Dis.* 2005; 193:503–507.
74. Raune D, Bebbington P, Dunn G, Kuipers E. Event attributes and the content of psychotic experiences in first-episode psychosis. *Psychol Med.* 2006;36:221–230.
75. Holmes EA, Steel C. Schizotypy: a vulnerability factor for traumatic intrusions. *J Nerv Ment Dis.* 2004;192:28–34.
76. Freeman D, Slater M, Bebbington PE, et al. Can virtual reality be used to investigate persecutory ideation? *J Nerv Ment Dis.* 2003;191:509–514.
77. Freeman D, Garety PA, Bebbington P, et al. The psychology of persecutory ideation II: a virtual reality experimental study. *J Nerv Ment Dis.* 2005;193:309–315.
78. Beck AT. Successful outpatient psychotherapy of a chronic schizophrenic with a delusion based on borrowed guilt. *Psychiatry*. 1952;15:305–312.
79. Pilling S, Bebbington P, Kuipers E, et al. Psychological treatments in schizophrenia: I meta-analysis of family intervention and CBT. *Psychol Med.* 2002;32:763–782.
80. Tarrier N, Wykes T. Is there evidence that cognitive behaviour therapy is an effective treatment for schizophrenia? A cautious or cautionary tale? *Behav Res Ther.* 2004;42:1377–1401.
81. Jones C, Cormac I, Silveira Da Mota Neto JI, Campbell C. Cognitive behaviour therapy for schizophrenia. *Cochrane Database Syst Rev.* 2005;4:1–57.
82. Zimmermann G, Favrod J, Trieu VH, Pomini V. The effect of cognitive behavioural treatment on the positive symptoms of schizophrenia spectrum disorders: a meta-analysis. *Schizophr Res.* 2005;77:1–9.
83. Lewis S, Tarrier N, Haddock G, et al. Randomised controlled trial of cognitive-behavioural therapy in early schizophrenia: acute-phase outcomes. *Br J Psychiatry*. 2002; 181(suppl 43):s91–s97.
84. Morrison AP, French P, Walford L, et al. Cognitive therapy for the prevention of psychosis in people at ultra-high risk. Randomised controlled trial. *Br J Psychiatry*. 2004;185: 291–297.
85. Craig TKJ, Garety P, Power P, et al. The Lambeth Early Onset (LEO) team: randomized controlled trial of the effectiveness of specialized care for early psychosis. *BMJ.* 2004;329:1067.
86. Kuipers E, Holloway F, Rabe-Hesketh S, Tennakoon L. An RCT of early intervention in psychosis: Croydon Outreach and Assertive Support Team (COAST). *Soc Psychiatry Psychiatr Epidemiol*. 2004;39:358–363.
87. Garety PA, Craig TKJ, Dunn G, et al. Specialised care for early psychosis: symptoms, social functioning and patient satisfaction: randomised controlled trial. *Br J Psychiatry*. 2006;188:37–45.
88. *NICE Guidelines for Psychological Treatment in Schizophrenia.* London, England: Gaskell Press; 2003.
89. Fowler D, Garety P, Kuipers E. *Cognitive Behaviour Therapy for People with Psychosis.* East Sussex, England: Wiley; 1995.
90. Chadwick P, Birchwood M, Trower P. *Cognitive Therapy for Delusions, Voices and Paranoia.* West Sussex, England: Wiley; 1996.
91. Kingdon DG, Turkington D. *The Case Study Guide to Cognitive Behaviour Therapy of Psychosis.* Chichester, UK: John Wiley & Sons; 2002.
92. Kingdon DG, Turkington D. *Cognitive Therapy of Schizophrenia: Guides to Evidence-Based Practice.* New York, NY: Guilford; 2005.
93. Turkington D, Kingdon D, Weiden PJ. Cognitive behaviour therapy for schizophrenia. *Am J Psychiatry*. 2006;163: 365–373.
94. Kuipers E, Fowler D, Garety P, et al. The London East Anglia randomised controlled trial of cognitive behaviour therapy for psychosis III: follow up and economic evaluation at 18 months. *Br J Psychiatry*. 1998;173:69–74.
95. Fowler D, Freeman D, Steel C, et al. The catastrophic interaction hypothesis: how does stress, trauma, emotion and information processing abnormalities lead to psychosis. In: Morrison A, Larkin W, eds. *Trauma and Psychosis.* John Wiley and Sons: In press.
96. Myin-Germeys I, Krabbendam L, Delespaul PAEG, Van Os J. Do life events have their effect on psychosis by influencing the emotional reactivity to daily life stress? *Psychol Med.* 2003;33:327–333.
97. Addington J, Burnett P. Working with families in the early stages of psychosis. In: Gleeson JFM, McGorry PD, eds. *Psychological Interventions for Early Psychosis.* Chichester, UK: Wiley and Sons; 2004.
98. Kuipers E, Leff J, Lam D. *Family Work for Schizophrenia: A Practical Guide.* 2nd ed. London, England: Gaskell Press; 2002.
99. Barrowclough C, Tarrier N. *Families of Schizophrenic Patients: Cognitive Behavioural Intervention.* London, England: Chapman & Hall; 1992.
100. Falloon IRH, Boyd JL, McGill CW. *Family Care of Schizophrenia.* New York, NY: Guilford Press; 1984.